Editorials

WJM Welcomes Arizona and Montana

THE ARIZONA MEDICAL ASSOCIATION and the Montana Medical Association have each voted to join the *WJM* family of sponsoring state medical societies. They join the medical associations of California, Idaho, Nevada, New Mexico, Utah, Washington and Wyoming (as well as six research and specialty societies) in designating *WJM* as their official scientific journal. The editors and staff of *WJM* welcome them and look forward to many years of shared endeavor.

The monthly circulation of *WJM* will now be something more than 53,000. It is worth noting that the subscribers and readers extend far beyond the boundaries of the western medical associations and societies that officially sponsor the journal. There are paid subscribers in every state of the union and most foreign countries. Reprint requests during the last year for the Epitomes section alone (the only section for which we are able to monitor this) have been received from Australia, Belgium, Bulgaria, Canada, Cuba, Czechoslovakia, East Germany, Ecuador, Estonia, France, Israel, Italy, Mexico, Nigeria, Poland, Romania, Spain, Venezuela and West Germany.

So it is clear that as the journal has been developing its strong base among the western states, it has not only been serving the members of its family of state medical associations and other affiliated societies, but has also begun to extend the influence of medicine in the West to the rest of the nation and to many far away parts of the world.

MSMW

The Mind, the Brain and Ulcer Disease

PEPTIC ULCER DISEASE in humans results from an imbalance between aggressive forces—acid and the proteolytic enzyme pepsin—and the endogenous defense mechanisms, which may include mucus and bicarbonate secretion, the maintenance of adequate mucosal blood flow and the inherent ability of the epithelium to maintain its integrity in the face of a large proton gradient. Since the range of acid-pepsin secretion is wide and most duodenal ulcer patients fall within the normal limits, the implication is that impaired mucosal resistance is the primary factor in ulcer formation. These defensive mechanisms are apparently influenced by both genetic and environmental factors, but quantitative assessment of these factors is not yet possible.

To perceive disease formation as the result of a simple imbalance of two opposing forces is of course reasonable, but simplistic. It would be equally simplistic to assume that an interruption of an intercontinental telephone conversation occurred because the plug was pulled on one of the telephones involved. Although it may be the case, the problem may well exist anywhere in the myriad of switches and computers that permit the conversation to occur.

In the symposium "Neurobiologic and Psychobiologic Mechanisms in Gastric Function and Ulceration" published in this issue of the journal, a group of scientists representing multiple disciplines review the data available on this subject. Although the symposium is scholarly in its scope, one is left with a sense of confusion, not from the presentation specifically, but from the unease one senses when confronted with many pieces of a gigantic, complex puzzle not yet organized.

Several questions contribute to this complexity. Are these experimental animal models relevant to human function and disease? Can the interspecies variations (cat, dog, rat) be reduced sufficiently to permit adequate conclusions? Can any of the numerous mechanisms noted in the symposium—the neural pathways, mechanoreceptors, chemoreceptors, osmoreceptors in the gastrointestinal tract and the liver and neuropeptides in the brain and gut—be shown to be physiologically relevant and not just demonstrable phenomena elicited in the experimental setting? Furthermore, to fully understand the mechanisms that control both feeding behavior and gastric function in humans, should we not search for the integrative links among (1) experience, that which occurs from the environment; (2) internal conflict—that is, unconscious processes of the mind—and (3) the neurophysiology?

As the discussants describe, the amygdalar structure within the limbic system has been implicated in emotionality and responsivity to stress. Neural connections between the central nucleus of the amygdala and the lateral hypothalamic area have been documented in rats.[1] Electrolytic lesions of the centromedial amygdaloid area result in marked alteration of gastric acid secretion. The lateral hypothalamus has, in addition to its connections with the limbic system, connections with the autonomic nervous system. The nature of these connections, however, is still unclear. The lateral hypothalamic area in various species has been shown to be clearly involved in the regulation of gastrointestinal function. Therefore, the evidence suggests that there is demonstrable "machinery" that may control gastrointestinal function existing well within the physical confines of the brain. In addition, in the symposium, Novin states that there "seems to be good evidence that the entire upper alimentary canal, the liver and the brain provide input important in feeding regulation." However, he goes on, "none are necessary components of that regulation," strongly suggesting "a redundant system of multiple inputs." Presumably, the brain integrates the diverse information "in a hierarchical organization," with increasingly complex afferent input and integration probably requiring higher levels of the nervous system.

However, it is not yet established how the *mind*—the anatomically elusive mental apparatus that is the source of both conscious and unconscious psychic processes—interacts with the physical system described in the symposium. What is the role of the mind in regard to gastric function and ulcer formation? There is some evidence that the mind does influence the rate of acid secretion and the motility of the stomach, but it is not yet established that this relationship is important in the cause of ulcer disease. Psychological stress has been considered to have an important role in either the genesis or exacerbation of ulcer disease. Here again, the evidence has been